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NEWS 8	AUG 18	FROSTI and KOSMET enhanced with Simultaneous Left and Righ Truncation
NEWS 9	AUG 18	Simultaneous left and right truncation added to ANABSTR
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NEWS 18	DEC 08	IMS file names changed
NEWS 19	DEC 09	Experimental property data collected by CAS now available in REGISTRY
NEWS 20	DEC 09	STN Entry Date available for display in REGISTRY and CA/CAplus
NEWS 21	DEC 17	DGENE: Two new display fields added
NEWS 22	DEC 18	BIOTECHNO no longer updated
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FILE 'HOME' ENTERED AT 08:05:05 ON 22 DEC 2003

=> file medline biosis embase caplus
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FILE 'MEDLINE' ENTERED AT 08:05:15 ON 22 DEC 2003

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=> s dimer (s) fusion (s) steroid (s) receptor
L1 9 DIMER (S) FUSION (S) STEROID (S) RECEPTOR

=> dup rem l1
PROCESSING COMPLETED FOR L1
L2 6 DUP REM L1 (3 DUPLICATES REMOVED)

=> d l2 total ibib kwic

L2 ANSWER 1 OF 6 EMBASE
on STN
ACCESSION NUMBER: 2000043997 EMBASE
TITLE: Interactions of the nuclear matrix-associated steroid receptor binding factor with its DNA binding element in the c-myc gene promoter.
AUTHOR: Barrett T.J.; Sandhu N.P.; Tomlinson A.J.; Benson L.M.; Subramaniam M.; Naylor S.; Spelsberg T.C.
CORPORATE SOURCE: T.C. Spelsberg, Dept. of Biochemistry/Molec. Biol., Mayo Clinic, 200 First Street, S.W., Rochester, MN 55905, United States. Spelsberg.thomas@mayo.edu
SOURCE: Biochemistry, (1 Feb 2000) 39/4 (753-762).
Refs: 76
ISSN: 0006-2960 CODEN: BICHAW
COUNTRY: United States
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 022 Human Genetics
029 Clinical Biochemistry
LANGUAGE: English
SUMMARY LANGUAGE: English
AB **Steroid receptor** binding factor (RBF) was originally isolated from avian oviduct nuclear matrix. When bound to avian genomic DNA, RBF generates saturable high-affinity binding sites for the avian progesterone **receptor** (PR). Recent studies have shown that RBF binds to a 54 bp element in the 5'- flanking region of the. . . paper, electrophoretic mobility shift assays (EMSAs) and S1 nuclease treatment are used to demonstrate that the RBF-maltose binding protei (MBP) **fusion** protein binds to single-stranded DNA of its element. Only the N-terminal domain of RBF binds the RBF DNA element as. . . support that the nuclear matrix binding site (acceptor site) for PR in the c-myc gene promoter is composed of RBF **dimers** bound to a specific single-stranded DNA element. The **dimers** of RBF are generated by C- terminal leucine zipper and the DNA binding occurs at the N-terminal parallel .beta.-sheet DNA. . .

L2 ANSWER 2 OF 6 EMBASE COPYRIGHT 2003 ELSEVIER INC. ALL RIGHTS RESERVED. on STN
ACCESSION NUMBER: 1998336916 EMBASE
TITLE: Studies of dehydroepiandrosterone (DHEA) with the human estrogen receptor in yeast.
AUTHOR: Nephew K.P.; Sheeler C.Q.; Dudley M.D.; Gordon S.; Nayfield S.G.; Khan S.A.
CORPORATE SOURCE: K.P. Nephew, Medical Sciences Program, Indiana University School Medicine, 302 Jordon Hall, Bloomington, IN 47405-4401, United States. knephew@indiana.edu
SOURCE: Molecular and Cellular Endocrinology, (25 Aug 1998) 143/1-2 (133-142).
Refs: 72
ISSN: 0303-7207 CODEN: MCEND6
PUBLISHER IDENT.: S 0303-7207(98)00128-2
COUNTRY: Ireland
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 003 Endocrinology
029 Clinical Biochemistry
LANGUAGE: English
SUMMARY LANGUAGE: English
AB Dehydroepiandrosterone (DHEA) is a C19 adrenal **steroid** synthesized in the human adrenal cortex and serving as a biosynthetic precursor to testosterone and 17.beta.-estradiol. Despite the fact that it is one of the most abundant **steroid** hormones in circulation, the physiological role of DHEA in humans remains unclear. The action of DHEA itself, such as its interactions with **receptors** and nuclear transcription factors, is not well understood, and a specific DHEA **receptor** has yet to be identified. Although the activity of DHEA can be due to its metabolism into androgens and estrogens, DHEA has been shown to interact with the androgen **receptor** and the estrogen **receptor** (ER) in vitro. We demonstrate in this study that DHEA (3.beta.-Hydroxy-5.alpha. -androstan-17-one) inhibits 17.beta.-estradiol (E2) binding to its **receptor** in vivo in yeast. DHEA stimulates human ER dimerization in yeast, as determined by ER **fusion** protein interactions, GAL4 reconstitution and subsequent measurement of increased .beta.-galactosidase activity. DHEA causes an increase in estrogen response element-dependent .beta.-galactosidase activity, demonstrating that the ER **dimer** induced by DHEA is transcriptionally active, but at a concentration of DHEA about 1000 times greater than E2. Inclusion of the nuclear **receptor** co-activator RIP140 in the yeast enhances ER transactivation by DHEA or E2 in a ligand-dependent manner; moreover, only in the presence of RIP140 is DHEA able to stimulate .beta.-galactosidase activity to levels similar to those achieved by E2. Ligand-**receptor** interaction for other C19-**steroids** was also examined. While 5-androstene-3.beta., 17.beta.-diol (ADIOL) displayed estrogenic activity in this system, 4-androstene-17-dione (androstenedione) and 4-androstene-17.beta.-o1,3-one (testosterone) did not.. . .

L2 ANSWER 3 OF 6 MEDLINE on STN DUPLICATE 1
ACCESSION NUMBER: 1998079032 MEDLINE
DOCUMENT NUMBER: 98079032 PubMed ID: 9417052
TITLE: Intermolecular NH2-/carboxyl-terminal interactions in androgen receptor dimerization revealed by mutations that cause androgen insensitivity.
AUTHOR: Langley E; Kemppainen J A; Wilson E M
CORPORATE SOURCE: Laboratories for Reproductive Biology, University of North Carolina, Chapel Hill, North Carolina 27599, USA.
CONTRACT NUMBER: HD16910 (NICHD)
IU54-HD35041 (NICHD)
P30-HD18968 (NICHD)
SOURCE: JOURNAL OF BIOLOGICAL CHEMISTRY, (1998 Jan 2) 273 (1)

92-101.
Journal code: 2985121R. ISSN: 0021-9258.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199802
ENTRY DATE: Entered STN: 19980217
Last Updated on STN: 19980217
Entered Medline: 19980203

AB Structural alignment of the human androgen **receptor** **dimer** was investigated by introducing **steroid** binding domain mutations that cause partial or complete androgen insensitivity into **fusion** proteins containing the full-length androgen **receptor** or the **steroid** binding domain. Most of the mutants had unchanged apparent equilibrium androgen binding affinity and increased dissociation rates of [3H]methyltrienolone and. . .

L2 ANSWER 4 OF 6 EMBASE COPYRIGHT 2003 ELSEVIER INC. ALL RIGHTS RESERVED. on STN
ACCESSION NUMBER: 95255569 EMBASE
DOCUMENT NUMBER: 1995255569
TITLE: The monomer-binding orphan receptor Rev-Erb represses transcription as a dimer on a novel direct repeat.
AUTHOR: Harding H.P.; Lazar M.A.
CORPORATE SOURCE: Univ. of Pennsylvania School of Med., Department of Medicine, 415 Curie Blvd.,Philadelphia, PA 19104-6149, United States
SOURCE: Molecular and Cellular Biology, (1995) 15/9 (4791-4802). ISSN: 0270-7306 CODEN: MCEBD4
COUNTRY: United States
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 029 Clinical Biochemistry
LANGUAGE: English
SUMMARY LANGUAGE: English

AB Rev-Erb is an orphan nuclear **receptor** which binds as a monomer to the thyroid/retinoic acid **receptor** half-site AGGTCA flanked 5' by an A/T-rich sequence, referred to here as a Rev monomer site. **Fusion** of Rev-Erb to the DNA binding domain of yeast GAL4 strongly repressed basal transcription of a GAL4-luciferase reporter gene as. . . binding site selection strategy was devised to test the hypothesis that Rev-Erb may function on a different site as a **dimer**. This approach identified sequences containing two Rev monomer sites arranged as direct repeats with the AGGTCA motifs separated by 2. . . this repression, consistent with the GAL4 results. However, the Rev-DR2 specificity did not require the C terminus in vivo, since **fusion** of C-terminally truncated Rev-Erb to a heterologous transactivation domain created a transcriptional activator specific for Rev-DR2. In addition to idealized. . . as retinoic acid-induced transcription from a naturally occurring Rev-DR2 in the CRBPI gene. Thus, although Rev-Erb is distinguished from other thyroid/**steroid** **receptor** superfamily members by its ability to bind DNA as a monomer, it functions as a homodimer to repress transcription of. . .

L2 ANSWER 5 OF 6 EMBASE COPYRIGHT 2003 ELSEVIER INC. ALL RIGHTS RESERVED. on STN
ACCESSION NUMBER: 94311345 EMBASE
DOCUMENT NUMBER: 1994311345
TITLE: Dimerization characteristics of the DNA- and steroid-binding domains of the androgen receptor.
AUTHOR: Nemoto T.; Ohara-Nemoto Y.; Shimazaki S.; Ota M.
CORPORATE SOURCE: Department of Biochemistry, Iwate Medical Univ. School Dentistry, Morioka,Iwate 020, Japan
SOURCE: Journal of Steroid Biochemistry and Molecular Biology,

(1994) 50/5-6 (225-233).
ISSN: 0960-0760 CODEN: JSBBEZ
COUNTRY: United Kingdom
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 003 Endocrinology
029 Clinical Biochemistry
LANGUAGE: English
SUMMARY LANGUAGE: English
AB The DNA-binding domain (DBD) of the androgen, mineralocorticoid, and glucocorticoid **receptors** and the **steroid**-binding domain (SBD) of the androgen **receptor** (AR) were expressed separately as **fusion** proteins with glutathione-S-transferase (GST) in Escherichia coli. Native polyacrylamide gel electrophoresis and gel exclusion HPLC demonstrated that the GST-ARDBD **fusion** protein was present as a **dimer**. On the other hand, the GST-ARSBD **fusion** protein formed a high-molecular weight oligomer, which seemed to be formed by two separate interactions, i.e. GST-GST and ARSBD-ARSBD between the **fusion** molecules. These findings strongly suggest that ARSBD has a potent ability to form a homodimer and that ARDBD does not.. . . specifically interacted with the glucocorticoid response elements of the mouse mammary tumor virus long terminal repeat (GRE(MMTV)). Cleavage of the **fusion** protein by thrombin abolished the binding, while the nonspecific DNA-cellulose binding ability was retained. Therefore, the dimeric configuration of GST-ARDBD,. . . apparent different in the binding affinity to these response elements was observed among the DBDs of androgen, mineralocorticoid and glucocorticoid **receptors**.

L2 ANSWER 6 OF 6 EMBASE COPYRIGHT 2003 ELSEVIER INC. ALL RIGHTS RESERVED. on STN
ACCESSION NUMBER: 89126316 EMBASE
DOCUMENT NUMBER: 1989126316
TITLE: Cooperative binding of steroid hormone receptors contributes to transcriptional synergism at target enhancer elements.
AUTHOR: Tsai S.Y.; Tsai M.-J.; O'Malley B.W.
CORPORATE SOURCE: Department of Cell Biology, Baylor College of Medicine, Houston, TX 77030, United States
SOURCE: Cell, (1989) 57/3 (443-448).
ISSN: 0092-8674 CODEN: CELLB5
COUNTRY: United States
DOCUMENT TYPE: Journal
FILE SEGMENT: 003 Endocrinology
029 Clinical Biochemistry
LANGUAGE: English
SUMMARY LANGUAGE: English
AB We demonstrated previously that two molecules of **steroid** hormone **receptor** bound efficiently to a single hormone response element (GRE/PRE) of the tyrosine aminotransferase gene (Tsai et al., 1988). Here, we show that wo tandemly linked GRE/PREs conferred progesterone inducibility synergistically to a heterologous TK-CAT **fusion** gene. Binding studies demonstrated that occupation of one GRE/PRE site by a progesterone **receptor dimer** increased the binding affinity of **receptors** for the second GRE/PRE site 100-fold. Thus, the observed synergistic induction of TK-CAT may result from cooperative binding of **receptor dimers** to the two GRE/PRE sites.

=> d his

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FILE 'MEDLINE, BIOSIS, EMBASE, CAPLUS' ENTERED AT 08:05:15 ON 22 DEC 2003

```
L1             9 S DIMER (S) FUSION (S) STEROID (S) RECEPTOR
L2             6 DUP REM L1 (3 DUPLICATES REMOVED)

=> s dimer (s) fusion (s) nuclear (s) hormone (s) receptor
L3             4 DIMER (S) FUSION (S) NUCLEAR (S) HORMONE (S) RECEPTOR

=> dup rem l3
PROCESSING COMPLETED FOR L3
L4             4 DUP REM L3 (0 DUPLICATES REMOVED)

=> d l4 total ibib kwic

L4   ANSWER 1 OF 4  EMBASE  COPYRIGHT 2003 ELSEVIER INC. ALL RIGHTS RESERVED.
     on STN
ACCESSION NUMBER:   2001422932  EMBASE
TITLE:              Domain structure of the NRIF3 family of coregulators
                    suggests potential dual roles in transcriptional
                    regulation.
AUTHOR:             Li D.; Wang F.; Samuels H.H.
CORPORATE SOURCE:   H.H. Samuels, Department of Pharmacology, Division of
                    Clinical Endocrinology, New York Univ. School of Medicine,
                    550 First Ave., New York, NY 10016, United States.
                    herbert.samuels@med.nyu.edu
SOURCE:             Molecular and Cellular Biology, (2001) 21/24 (8371-8384).
                    Refs: 63
                    ISSN: 0270-7306  CODEN: MCEBD4
COUNTRY:            United States
DOCUMENT TYPE:      Journal; Article
FILE SEGMENT:       029     Clinical Biochemistry
LANGUAGE:           English
SUMMARY LANGUAGE:   English
AB   The identification of a novel coregulator for nuclear
     hormone receptors, designated NRIF3, was recently
     reported (D. Li et al., Mol. Cell. Biol. 19:7191-7202, 1999). Unlike most
     known coactivators, NRIF3 exhibits a distinct receptor
     specificity in interacting with and potentiating the activity of only TRs
     and RXRs but not other examined nuclear receptors.
     However, the molecular basis underlying such specificity is unclear. In
     this report, we extended our study of NRIF3-receptor
     interactions. Our results suggest a bivalent interaction model, where a
     single NRIF3 molecule utilizes both the C-terminal LXXIL (receptor
     -interacting domain 1 [RID1]) and the N-terminal LXXLL (RID2) modules to
     cooperatively interact with TR or RXR (presumably a receptor
     dimer), with the spacing between RID1 and RID2 playing an
     important role in influencing the affinity of the interactions. During
     the. . . 112), which is predicted to form a coiled-coil structure and
     contains a putative leucine zipper, like motif. By using Gal4
     fusion constructs, we identified an autonomous transactivation
     domain (AD1) at the C terminus of NRIF3. Somewhat surprisingly,
     full-length NRIF3 fused to. . . additional isoforms due to alternative
     splicing. These two isoforms contain the same RepD1 region as NRIF3.
     Consistent with this, Gal4 fusions of these two isoforms were
     also found to repress transcription. Cotransfection of NRIF3 or its two
     isoforms did not relieve the transrepression function mediated by their
     corresponding Gal4 fusion proteins, suggesting that the
     repression involves a mechanism(s) other than the recruitment of a
     titratable corepressor. Interestingly, a single amino. . .

L4   ANSWER 2 OF 4  EMBASE  COPYRIGHT 2003 ELSEVIER INC. ALL RIGHTS RESERVED.
     on STN
ACCESSION NUMBER:   1999087786  EMBASE
TITLE:              A functional DNA binding domain is required for growth
                    hormone-induced nuclear accumulation of Stat5B.
AUTHOR:             Herrington J.; Ruin L.; Luo G.; Yuo-Lee L.-Y.; Carter-Su C.
```

CORPORATE SOURCE: C. Carter-Su, Dept. of Physiology, Univ. of Michigan Medical School, 6804 Medical Science II, 1301 Catherine St., Ann Arbor, MI 48109-0622, United States. cartersu@umich.edu
SOURCE: Journal of Biological Chemistry, (19 Feb 1999) 274/8 (5138-5145).
Refs: 49
ISSN: 0021-9258 CODEN: JBCHA3
COUNTRY: United States
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 029 Clinical Biochemistry
LANGUAGE: English
SUMMARY LANGUAGE: English

AB . . . regulating the cellular distribution of STAT family transcription factors remain poorly understood. To identify regions of StatSB required for ligand-induced **nuclear** accumulation, we constructed a cDNA encoding green fluorescent protein (GFP) fused to the N terminus of Stat5B and performed site-directed mutagenesis. When co-expressed with growth **hormone** (GH) **receptor** in COS-7 cells, GFP-StatSB is tyrosylphosphorylated, forms **dimers**, and binds DNA in response to GH in a manner indistinguishable from untagged Stat5B. In multiple cell types, laser scanning confocal imaging of GFP-Stat5B co-expressed with GH **receptor** shows that GFP-Stat5B undergoes a rapid, dramatic accumulation in the nucleus upon GH stimulation. We introduced alanine substitutions in several regions of Stat5B and assayed for GH-dependent **nuclear** localization. Only the mutation that prevented binding to DNA (466VVVI469) abrogated GH-stimulated **nuclear** localization. This mutant **fusion** protein is tyrosyl-phosphorylated and dimerizes in response to GH. These results suggest that either high affinity binding to DNA contributes to **nuclear** accumulation of Stat5B or that this region is crucial for two functions, namely accumulation of Stat5B in the nucleus and DNA binding. Thus, we have identified a mutant Stat5 defective in **nuclear** localization despite its ability to be tyrosyl-phosphorylated and to dimerize.

L4 ANSWER 3 OF 4 EMBASE COPYRIGHT 2003 ELSEVIER INC. ALL RIGHTS RESERVED. on STN
ACCESSION NUMBER: 1999079975 EMBASE
TITLE: Identification of a nuclear localization signal in activin/inhibin -(A) subunit; intranuclear -(A) in rat spermatogenic cells.
AUTHOR: Blauer M.; Husgafvel S.; Syvala H.; Tuohimaa P.; Ylikomi T.
CORPORATE SOURCE: M. Blauer, Department of Anatomy, Medical School, University of Tampere, FIN-33101 Tampere, Finland. Blauer@csc.fi
SOURCE: Biology of Reproduction, (1999) 60/3 (588-593).
Refs: 50
ISSN: 0006-3363 CODEN: BIREBV
COUNTRY: United States
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 002 Physiology
028 Urology and Nephrology
029 Clinical Biochemistry
LANGUAGE: English
SUMMARY LANGUAGE: English

AB Activin is a dimeric glucoprotein **hormone** that was initially characterized by its ability to stimulate pituitary FSH secretion and was subsequently recognized as a growth factor. . . of tissues. In the testis, activin has been implicated in the auto/paracrine regulation of spermatogenesis through its cognate cell membrane **receptors** on Sertoli and germ cells. In this study we provide evidence for intranuclear activin/inhibin -(A) subunit and show its distribution in the rat seminiferous epithelium. We have shown by transient expression in HeLa cells of .beta.-galactosidase **fusion** proteins that the .beta.(A)

subunit precursor contains a functional **nuclear** localization signal within the lysine-rich sequence corresponding to amino acids 231-244. In all stages of the rat seminiferous epithelial cycle, an intense immunohistochemical staining of **nuclear** .beta.(A) was demonstrated in intermediate or type B spermatogonia or primary spermatocytes in their initial stages of the first meiotic. . . cytoplasm, suggesting disposal of .beta.(A) before spermatozoal maturation. Immunoblot analysis of a protein extract from isolated testicular nuclei revealed a **nuclear** .beta.(A) species with a molecular mass of approximately 24 kDa, which is more than 1.5 times that of the mature .beta.(A) subunit present in activin **dimers**. These results suggest that activin/inhibin .beta.(A) may elicit its biological functions through two parallel signal transduction pathways, one involving the dimeric molecule and cell surface **receptors** and the other an alternately processed .beta.(A) sequence acting directly within the nucleus. According to our immunohistochemical data, .beta.(A) may play a significant role in the regulation of **nuclear** functions during meiosis and spermiogenesis.

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ACCESSION NUMBER: 1998336916 EMBASE

TITLE: Studies of dehydroepiandrosterone (DHEA) with the human estrogen receptor in yeast.

AUTHOR: Nephew K.P.; Sheeler C.Q.; Dudley M.D.; Gordon S.; Nayfield S.G.; Khan S.A.

CORPORATE SOURCE: K.P. Nephew, Medical Sciences Program, Indiana University School Medicine, 302 Jordon Hall, Bloomington, IN 47405-4401, United States. knephew@indiana.edu

SOURCE: Molecular and Cellular Endocrinology, (25 Aug 1998) 143/1-2 (133-142).
Refs: 72
ISSN: 0303-7207 CODEN: MCEND6

PUBLISHER IDENT.: S 0303-7207(98)00128-2

COUNTRY: Ireland

DOCUMENT TYPE: Journal; Article

FILE SEGMENT: 003 Endocrinology
029 Clinical Biochemistry

LANGUAGE: English

SUMMARY LANGUAGE: English

AB . . . as a biosynthetic precursor to testosterone and 17.beta.-estradiol. Despite the fact that it is one of the most abundant steroid **hormones** in circulation, the physiological role of DHEA in humans remains unclear. The action of DHEA itself, such as its interactions with **receptors** and **nuclear** transcription factors, is not well understood, and a specific DHEA **receptor** has yet to be identified. Although the activity of DHEA can be due to its metabolism into androgens and estrogens, DHEA has been shown to interact with the androgen **receptor** and the estrogen **receptor** (ER) in vitro. We demonstrate in this study that DHEA (3.beta.-Hydroxy-5.alpha. -androstan-17-one) inhibits 17.beta.-estradiol (E2) binding to its **receptor** in vivo in yeast. DHEA stimulates human ER dimerization in yeast, as determined by ER **fusion** protein interactions, GAL4 reconstitution and subsequent measurement of increased .beta.-galactosidase activity. DHEA causes an increase in estrogen response element-dependent .beta.-galactosidase activity, demonstrating that the ER **dimer** induced by DHEA is transcriptionally active, but at a concentration of DHEA about 1000 times greater than E2. Inclusion of the **nuclear receptor** co-activator RIP140 in the yeast enhances ER transactivation by DHEA or E2 in a ligand-dependent manner; moreover, only in the presence of RIP140 is DHEA able to stimulate .beta.-galactosidase activity to levels similar to those achieved by E2. Ligand-**receptor** interaction for other C19-steroids was also examined. While 5-androstene-3.beta., 17.beta.-diol (ADIOL) displayed

estrogenic activity in this system, 4-androstene-17-dione (androstenedione) and. . .